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ENDOCRINE DISRUPTORS AND RISK ASSESSMENT: POTENTIAL FOR A BIG MISTAKE

KEITH J. JONES, ESQ.

I. INTRODUCTION

Trillions of dollars could be spent in an attempt to regulate endocrine disruptors as drinking water pollutants, and it could be a complete waste of money if the regulations are based on traditional environmental risk assessment. The federal government is expected to spend billions of dollars just to develop test methods to identify chemicals that are endocrine disruptors. Once endocrine disruptors can be identified and measured in water, traditional environmental risk assessment would attempt to determine the amount of harm caused by a specific level of an individual endocrine disruptor. This process could also cost billions of dollars. However, because low levels of one endocrine disruptor may combine with low levels of a different endocrine disruptor that acts in a similar fashion to cause harm, this approach may not be very effective. Nevertheless, this method could be the basis for government regulations requiring drinking water plants to reduce levels of individual endocrine disruptors at a staggering cost with little or no benefit to human health.

In an effort to thoroughly explore the issue stated above, this Article will review relevant historical and background materials regarding endocrine disruptors. It will then explain what endocrine disruptors are and how they are believed to work. Next, the Article will look at traditional environmental risk assessment. Following that, it will describe how water is generally regulated in the United States. The Article will then specifically describe the federal government's efforts to regulate radon as a drinking water pollutant. Finally, it will suggest that a similar regulatory approach that relies upon traditional risk assessment might be inadequate for regulating endocrine disrupting chemicals as drinking water pollutants because of their possibly cumulative effects.

II. HISTORY AND BACKGROUND

It is well established that certain chemicals can affect hormones. More than half a century ago, researchers observed that substances extracted from plants could imitate hormones when in-

jected into animals.¹ In the 1950s, it was shown that the insecticide DDT could feminize newborn roosters.² In her seminal work, *SILENT SPRING*, Rachel Carson warned against the possibly negative consequences of society's increasing exposure to a "wide variety of synthetic estrogens."³ Nevertheless, in the 1960s, manufactured hormones that mimicked estrogen and progestin began to be mass marketed to the public in the form of birth control pills. By the end of the 1970s, it was determined that certain pesticides were the probable cause of sterility in men who had worked with them and that various chemicals could reduce sperm counts.⁴

During the 1980s, a growing number of scientists documented cases of wildlife suffering from an assortment of reproductive, developmental and behavioral abnormalities.⁵ No one, however, appeared to be seeing the big picture until 1987, when a grandmother with a recently-acquired Ph.D. in zoology started gathering hundreds of research papers about the Great Lakes in an effort to gauge pollution levels only to stumble upon a potentially more pervasive problem.⁶ Theo Colborn was a pharmacist and a shepherd before pursuing her doctorate later in life.⁷ Perhaps it was her unique background that enabled her to suspect connections related to hormonal disruption in the seemingly unrelated reports she reviewed. Regardless, Colborn's work led her to others who shared her suspicions, and in July 1991, she brought together several scientists from varying fields to the Wingspread Conference Center in Racine, Wisconsin to share and discuss their work related to hormone disruption.⁸

Amazingly, the twenty-one participants at the Wingspread Conference, whose specialties ran the gamut from anthropology to zo-

1. See BURNHAM WALKER & JAMES JANNEY, *ENDOCRINOLOGY* 389-92 (1930) (demonstrating how hormonal study has gone on for decades).

2. See H. Burlington & V.F. Lindeman, *Effect of DDT on Testes and Secondary Sex Characteristics of White Leghorn Cockerels*, *SOC'Y FOR EXPERIMENTAL BIOLOGY AND MED.*, 48-51 (1950) (naming specific instance of hormones altering animal).

3. See RACHEL CARSON, *SILENT SPRING* 235-37 (1962) (warning of dangers from synthetic estrogen exposure).

4. See DEVRA DAVIS, *WHEN SMOKE RAN LIKE WATER: TALES OF ENVIRONMENTAL DECEPTION AND THE BATTLE AGAINST POLLUTION* 193-222 (2002) (specifying reproductive damage from endocrine disruptors).

5. See THEO COLBORN ET AL., *OUR STOLEN FUTURE: HOW WE ARE THREATENING OUR FERTILITY, INTELLIGENCE AND SURVIVAL—A SCIENTIFIC DETECTIVE STORY* 6-10 (1996) [hereinafter COLBORN] (citing numerous studies on detrimental effect of endocrine disruptors on various species).

6. See *id.* at 11-12 (explaining accidental discovery of reproductive issues in animals).

7. See *id.* at 13 (citing background of woman who brought issues to light).

8. See *id.* at 170 (highlighting result of Colborn's efforts).

ology, were able to reach a consensus in just three days.⁹ Their Consensus Statement begins as follows:

We are certain of the following: A large number of man-made chemicals that have been released into the environment, as well as a few natural ones, have the potential to disrupt the endocrine system of animals, including humans. Among these are the persistent, bioaccumulative, organohalogen compounds that include some pesticides (fungicides, herbicides, and insecticides) and industrial chemicals, other synthetic products, and some metals.¹⁰

The “endocrine disrupting hypothesis,” or claim that human health is threatened by exposure to a multiplicity of chemicals that interfere with the body’s hormone system, was brought to the attention of the general public in 1996 when Colborn co-authored a best-selling novel, *OUR STOLEN FUTURE*. The novel reads like a detective story and chronicles Colborn’s years of piecing together clues of hormone disruption she found in the reports of other scientists from divergent fields of study.

Around the same time that Colborn was discovering the world of hormone disruption, a group of breast cancer activists from New York City were heavily lobbying the United States Congress to fund cancer research.¹¹ They believed that the release of synthetic chemicals into the environment had resulted in an increase in the incidence of breast cancer on Long Island, New York.¹² A Long Island breast cancer study suggested that “estrogen metabolism” was possibly a vehicle for allowing organochlorine compounds to cause breast cancer.¹³ Consequently, New York Senator Alfonse D’Amato took the lead in obtaining over one million dollars of funding for breast cancer research and eventually calling for the

9. See *id.* (demonstrating clear necessity of solution by clear agreement).

10. See COLBORN, *supra* note 5, at 260-61 (quoting part of Wingspread Conference Consensus Statement).

11. See Linda Saslow, *Transformed by Passion and Politics, Breast Cancer Movement Gains Power*, N.Y. TIMES, Nov. 8, 1998, at 14LI (introducing another group investigating cancer).

12. See *id.* (mentioning possible link between hormonal changes and breast cancer).

13. See *Major Study Scrutinizes Environmental Carcinogens*, PUBLIC HEALTH MAGAZINE, Winter 1995, Vol. 5, No. 1, available at <http://www.cumc.columbia.edu/news/chronicle/archives/phmg0027.html> (discussing study of causes of Long Island cancer).

creation of endocrine disruptor screening and testing programs through federal legislation.¹⁴

In 1995, the United States Environmental Protection Agency (EPA) held a series of workshops to develop a plan for assessing the possible health risks of endocrine disruptors.¹⁵ In May 1996, the EPA hosted a stakeholder meeting on the issue of endocrine disruptors, which led the EPA to focus on screening and testing for endocrine disruptors.¹⁶ Later that same year, in August, the United States Congress enacted the Food Quality Protection Act (FQPA)¹⁷ and amended the Safe Drinking Water Act (SDWA)¹⁸ in part to address endocrine disruptors and the need for more research and the necessity of developing reliable screening and testing protocols.¹⁹ The FQPA obviously focuses on food but also specifically targets pesticides.²⁰ The amendments to the SDWA are broader, and in a section titled "Estrogenic Substances Screening Program," they state the following:

[I]n addition to the substances referred to in the . . . [FQPA] the Administrator [of EPA] may provide for testing under the screening program authorized by . . . [FQPA] of any other substance that may be found in sources of drinking water if the Administrator determines that a substantial population may be exposed to such substance.²¹

Furthermore, several other federal statutes contain provisions, which may eventually be used as justification for additional endocrine disruptor testing. The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)²² gives the EPA authority to require testing of many pesticides.²³ The federal Food, Drug, and Cosmetics Act (FDCA)²⁴ also gives the EPA the authority to require testing of

14. See Saslow, *supra* note 11, at 14LI (explaining how study results led to funding for research and screening).

15. See Endocrine Disruptor Screening Program, 63 Fed. Reg. 42,852 (Aug. 11, 1998) (citing results of federal investigation into endocrine disruptors).

16. See *id.* (explaining how report results fostered action).

17. 7 U.S.C. § 136 (1996).

18. 42 U.S.C. § 300j-17 (1994 & Supp. 111 1997).

19. See *id.* (citing where EPA is given power to screen for contaminants).

20. See Food Quality Protection Act of 1996, 21 U.S.C. § 346(a) (1994 & Supp. III 1997) (mentioning specific government action in response to EDSTAC).

21. See Safe Drinking Water Act Amendments of 1996, 42 U.S.C. § 300j-17 (1994 & Supp. III 1997) (detailing EPA procedures).

22. 7 U.S.C. § 136(a)(c)(2)(B) (1994).

23. See *id.* (citing where EPA is given power to require pesticide testing).

24. 21 U.S.C. § 346(a) (1994 & Supp. III 1997).

other pesticides plus chemicals that may have a cumulative effect with pesticides.²⁵ Finally, the federal Toxic Substances Control Act (TSCA)²⁶ authorizes the EPA to require testing of hazardous chemicals when there are specific exposure-based findings to support such testing.²⁷ In October 1996, the EPA created a federal advisory committee to examine the issue of endocrine disruptors in greater detail than anyone had previously.²⁸ The Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC) brought together representatives from drinking water providers, environmental groups, the EPA, other federal agencies, state agencies, public health organizations, different industries, additional stakeholders and various scientists to share information about endocrine disruptors.²⁹ EDSTAC's primary purpose was to advise the EPA with regard to designing an adequate and reliable screening and testing program for endocrine disruptors.³⁰ In August 1998, EDSTAC issued its Final Report, which contained a plethora of recommendations. Notably, Congress had already required the EPA to develop a screening program for endocrine disruptors by August 1998.³¹ As a result, the EPA's Endocrine Disruptor Screening Program (EDSP) was published in the Federal Register on August 11, 1998.³² The EDSP was based on many of the recommendations made in the EDSTAC Final Report.

In addition to developing a screening program by 1998, Congress also required the EPA to implement such a screening program by 1999 and make a progress report to Congress regarding the program by 2000.³³ Nevertheless, the Natural Resources De-

25. See *id.* (mentioning where EPA is given power to test for chemicals that may have cumulative effect).

26. 15 U.S.C. § 2603 (1994).

27. See *id.* (exploring where EPA gets power to test hazardous chemicals).

28. See Endocrine Disruptor Screening Program, *supra* note 15, at 1-2 (citing reason for creation of EDSTAC).

29. See *id.* (detailing participants in EDSTAC).

30. See *id.* (stating purpose of EDSTAC).

31. See Food Quality Protection Act of 1996, 21 U.S.C. § 346(a)(1) (1994 & Supp. III 1997) (detailing general requirements for tolerance or exemption for pesticide chemical residues).

32. See Endocrine Disruptor Screening Program, *supra* note 15, at 1-2 (setting forth screening program to determine which pesticide chemicals and other substances may have effect in humans similar to effect produced by naturally occurring estrogen or other endocrine effects).

33. See 21 U.S.C. § 346(a)(2) (detailing requirements of chemical residues in processed food); Safe Drinking Water Act Amendments of 1996, 42 U.S.C. § 300j-18 (1994 & Supp. III 1997) (discussing drinking water studies identifying groups within general population who have adverse health effects from exposure to contaminants in drinking water).

fense Council (NRDC) ultimately filed an action against the EPA for failure to implement the EDSP in a timely manner.³⁴ The resulting consent decree memorialized a settlement agreement requiring the EPA to use “best efforts” to implement screening of certain suspected endocrine disruptors by specific dates.³⁵ In August 2000, the EPA did provide a progress report to Congress explaining the issue of possible endocrine disruption, describing the EDSP and the EPA’s efforts to implement it.³⁶

In October 2001, the EPA established the Endocrine Disruptor Method Validation Subcommittee (EDMVS) to oversee future implementation of the EDSP.³⁷ In May 2002, the EPA made another progress report to Congress summarizing the endocrine disruptor work it had done up to that point and the oversight of the EDMVS, particularly in the area of developing validation processes for new endocrine disruptor screening and testing methods.³⁸ In December 2002, the EPA published its “Proposed Chemical Selection Approach for Initial Round of Screening,” which is the method the EPA planned to use to select the first group of chemicals to be subjected to the EDSP.³⁹ In December 2003, the EPA’s Office of Re-

34. See *Natural Resources Defense Council v. Whitman*, 2001 U.S. Dist. LEXIS 18435, **3-4 (N.D. Cal. 2001) (complaining EPA did not adopt proper screening programs required by FQPA and failed to validate tests used in endocrine disruptor screening program by arbitrarily subjecting tests that did not use laboratory animals to more rigorous validation process than tests involving animals).

35. See *id.* at *10 (proposing settlement with three components: (1) consent decree binding EPA to time table for promulgating pesticide regulations; (2) voluntary dismissal of cause of action regarding endocrine-disruptor screening program and petition for review in Ninth Circuit; (3) entering private settlement agreement to make best efforts to implement endocrine-disruptor screening program that had been proposed by advisory committee formed by EPA, EDSTAC). NRDC objected to the settlement because it would harm its interests. See *id.*

36. See U.S. ENVIRONMENTAL PROTECTION AGENCY, ENDOCRINE DISRUPTOR SCREENING PROGRAM REPORT TO CONGRESS (Aug. 2000) (setting forth pertinent issues concerning endocrine disruptors including overview of endocrine disruptor issue, screening program, implementation process of endocrine disruptor research, alternative test method development, findings and recommendations for further testing and action).

37. See Endocrine Disruptor Method Validation Subcommittee, 66 Fed. Reg. 51,951, 51,151 (Oct. 11, 2001) (providing technical advice on Tier 1 Screening and Tier 2 Testing methods for EPA’s EDSP).

38. See U.S. ENVIRONMENTAL PROTECTION AGENCY, REPORT TO U.S. HOUSE OF REPRESENTATIVES COMMITTEE ON APPROPRIATIONS ON THE STATUS OF THE ENDOCRINE DISRUPTOR METHODS VALIDATION SUBCOMMITTEE (May 2002) (giving background and status of Endocrine Disruptor Methods Validation Subcommittee meetings and progress). It reported that upcoming meetings would discuss two tier screening process of laboratory methods performed with various animal assays. See *id.*

39. See Endocrine Disruptor Screening Program, Proposed Chemical Selection Approach for Initial Round of Screening; Request Comment, 67 Fed. Reg.

search and Development (ORD) issued a multi-year plan outlining its proposed course of action regarding endocrine disruptors through 2012.⁴⁰ The ORD's long-term objectives are to achieve a better understanding of endocrine disruptors, determine their impact on the environment, including humans, and support the EPA's screening and testing work.⁴¹

Toward the end of 2004, endocrine disruption became a popular story in the news. In October, the DENVER POST reported a story about a biologist who suspected the deformed fish he was finding in the South Platter River resulted from endocrine disruptors being discharged from a wastewater treatment plant upstream.⁴² In November, the PHILADELPHIA INQUIRER published a piece about birth control hormones and other possible endocrine disrupting chemicals being detected in drinking water.⁴³ In December, the Associated Press released an article suggesting endocrine disruptors were causing male fish in the Potomac River near Sharpsburg, Maryland to grow eggs.⁴⁴ As a result of such growing media coverage, it is

79,611 (Dec. 30, 2002) (selecting group of chemicals to be screened in Agency's EDSP but anticipating modification of chemical selection approach for Tier 1 screening lists after examining results of chemical tests, feasibility of incorporating different categories of chemicals and additional pathways to exposure, and availability of new priority-setting tools). The EPA developed the EDSP "to determine whether certain substances may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen, or such other effects as EPA may designate." See *id.* at 79,613. The statute requires the EPA to "provide for the testing of all pesticide chemicals" and gives the EPA discretionary authority to "provide for the testing of any other substance that may have an effect that is cumulative to an effect of a pesticide chemical if the Administrator determines that a substantial population may be exposed to such a substance." See *id.* The EPA also has discretionary authority to test "any other substances that may be found in sources of drinking water if the Administrator determines that a substantial population may be exposed to such substance." See *id.* at 79,614.

40. See OFFICE OF RESEARCH AND DEVELOPMENT, U.S. ENVIRONMENTAL PROTECTION AGENCY MULTI-YEAR PLAN (FY2000-2012) FOR ENDOCRINE DISRUPTORS (Dec. 2003) [hereinafter MULTI-YEAR PLAN] (identifying research already performed and future research needed in order to satisfy three specific long-term goals).

41. See *id.* at 10-13 (detailing research highlights corresponding to long-term goals).

42. See Theo Stein & Miles Moffeit, *Mutant Fish Prompt Concern: Study Focuses on Sewage Plant*, DENVER POST, Oct. 3, 2004, at A-01 (noting impact of endocrine disruptors on various animals in distinct areas with specific result of heightened female populations).

43. See Dawn Fallik, *Drinking Water Holds Surprises*, PHILA. INQUIRER, Nov. 28, 2004, <http://www.philly.com/mld/philly/living/health/10287666.htm> (discussing studies performed in New Jersey and Philadelphia that water filtration was not eliminating chemicals as previously believed).

44. See *Male Fish Growing Eggs Found in Potomac*, ASSOCIATED PRESS, Dec. 21, 2004 (reporting scientists found male fish growing eggs in Potomac River which could result from pollutants in water acting as endocrine disruptors).

likely that the public may soon demand regulation of endocrine disruptors rather than just screening and testing.

III. WHAT ARE ENDOCRINE DISRUPTING COMPOUNDS?

To understand what endocrine disrupting compounds (EDCs) are, one must first know a little about the endocrine system. Most people are probably more familiar with the term “hormone,” but it is synonymous with the term “endocrine” when used with the word “system.” Endocrine simply means secreting internally.⁴⁵ The purpose of the endocrine system or hormone system is to regulate an organism’s physical processes.⁴⁶ For example, the endocrine system regulates an organism’s growth.⁴⁷ The endocrine system has three basic parts: the glands, the hormones and the receptors.⁴⁸ The glands secrete hormones which travel through the bloodstream in search of the receptors.⁴⁹ Receptors are the part of a cell that binds with the hormone.⁵⁰ The reaction from this binding results in the hormone’s effect, such as stimulating growth.⁵¹ The hormone to receptor relationship is often described as a key to a lock relationship. Hormones are keys that travel throughout an organism’s entire body via the blood, but they should only affect cells that have the proper receptors, or correct locks.⁵²

Every fish, bird and mammal, including humans, has an endocrine system. The endocrine glands in the human body are the hypothalamus, the pituitary gland, the thyroid gland, the adrenal glands, the pancreas and the gonads.⁵³ The hypothalamus makes an essential connection between the endocrine system and the nervous system.⁵⁴ The pituitary gland is sometimes referred to as the “master gland” because through its secretions it essentially controls the thyroid, adrenals and gonads.⁵⁵ The thyroid gland modulates

45. See WEBSTER’S NINTH NEW COLLEGIATE DICTIONARY, 410-11 (9th ed. 1983) (defining endocrine).

46. See GEORGE HEDGE ET AL., CLINICAL ENDOCRINE PHYSIOLOGY 3-5 (1987) (analyzing endocrine physiology).

47. See *id.* at 317 (discussing functions of endocrine system).

48. See *id.* (noting various parts of endocrine system).

49. See *id.* at 3-5 (noting role of glands within endocrine system).

50. See *id.* (describing physiological parts and their role in hormonal process).

51. See HEDGE ET AL., *supra* note 46, at 322 (illustrating process through which endocrine system affects body function).

52. See *id.* (noting discussion process through which hormones attach to receptors).

53. See H. MAURICE GOODMAN, BASIC MEDICAL ENDOCRINOLOGY (Raven Press 1994) (noting parts of human endocrine system).

54. See *id.* at 29 (discussing role of hypothalamus within endocrine system).

55. See *id.* at 28 (noting function of pituitary gland).

the metabolism and affects growth and development.⁵⁶ The adrenals or adrenal glands produce hormones in reaction to stress and influence general cell function.⁵⁷ The pancreas produces the hormone's insulin and glucagon which regulate the levels of glucose or sugar in the blood.⁵⁸ The gonads produce steroids including androgen, estrogen and progestin, which all affect reproduction, development and growth.⁵⁹ Obviously, a well functioning endocrine system is critical for proper development and maintenance of a human body.

Generally speaking, endocrine disruptors are natural or synthetic chemicals including some pharmaceuticals, pesticides, industrial byproducts and combinations of such substances both natural and synthetic that have a disruptive effect on an organism's endocrine system.⁶⁰ After much debate, the EDSTAC defined an endocrine disruptor as "an exogenous chemical substance or mixture that alters the structure or function(s) of the endocrine system and causes adverse effects at the level of the organism, its progeny, populations, or sub-populations of organisms, based on scientific principles, data, weight of evidence, and the precautionary principle."⁶¹ Similarly, the ORD defined endocrine disruptors as "exogenous agents that interfere with the production, release, transport, metabolism, binding, action, or elimination of the natural hormones in the body responsible for the maintenance of homeostasis and the regulation of developmental processes."⁶² In both of these definitions, the term "exogenous" simply means from outside the organism.⁶³ Some feel the term "endocrine disruptor" is too inflammatory and have suggested the more neutral label of hormon-

56. See *id.* at 46 (describing utility of thyroid gland).

57. See *id.* at 51 (illustrating role of adrenal glands).

58. See GOODMAN, *supra* note 53, at 113 (describing role of pancreas as part of endocrine system).

59. See *id.* at 249, 271 (noting function of gonads).

60. See RETHA NEWBOLD & WENDY JEFFERSON, *Developmental and Reproductive Abnormalities Associated with Environmental Estrogens: Diethylstilbestrol (DES) as an Example*, in ENDOCRINE DISRUPTORS: EFFECTS ON MALE AND FEMALE REPRODUCTIVE SYSTEMS (CRC Press, 2005) (describing endocrine disruptors and their effect on endocrine system).

61. See EDSTAC Final Report, 63 Fed. Reg. 154, at 3-4 (Aug. 11, 1998) (codified at 40 C.F.R. pt. 180) (explaining EDSTAC definition of endocrine disruptor).

62. See MULTI-YEAR PLAN, *supra* note 40, at 4 (noting OPD definition of endocrine disruptor).

63. See WEBSTER'S NINTH NEW COLLEGIATE DICTIONARY 435 (9th ed. 1983) (defining "exogenous" as used in OPD and EDSTAC definition of endocrine disruptor).

ally active agents (HAA).⁶⁴ Nevertheless, probably partially due to its neutrality, HAA has not gained widespread acceptance by scientists, nor has the media embraced it.

Regardless of the mechanism's name, disruption of the endocrine system can occur in several different ways. Endocrine disruptors can mimic naturally occurring hormones. For example, the insecticide DDT is known to cause the same response in cells and tissues as naturally produced estrogen.⁶⁵ Exposure to endocrine disruptors that act like mimics can cause an organism's endocrine system to over-produce natural hormones that interact with receptors and result in various deviations, such as abnormal growth.⁶⁶ Still other endocrine disruptors only inhibit an organism's endocrine system and result in the underproduction of necessary hormones, limiting development or reproduction.⁶⁷ Finally, other endocrine disruptors can completely block the instructions from a hormone to its designated receptor, thereby altogether preventing normal endocrine function.⁶⁸

Just as the forms of endocrine disruption vary, so do the consequences. For years, biologists and ecologists have been documenting the harmful effects of endocrine disruptors on the reproduction and development of wildlife.⁶⁹ These effects include, but are not limited to: "eggshell thinning[;] population declines[;] impaired viability of offspring[;] altered hormone concentrations[;] and changes in sociosexual behavior."⁷⁰ Many of these effects have been subsequently duplicated in laboratory experiments

64. See COMMITTEE ON HORMONALLY ACTIVE AGENTS IN THE ENVIRONMENT, NATIONAL RESEARCH COUNCIL, HORMONALLY ACTIVE AGENTS IN THE ENVIRONMENT, 21 (1999) [hereinafter HORMONALLY ACTIVE AGENTS] (explaining contention with team "endocrine disruptor").

65. See Andrew Rooney & Louis Guillette, Jr., *Contaminant Interactions with Steroid Receptors: Evidence of Receptor Binding in* ENDOCRINE DISRUPTING CONTAMINANTS: AN EVOLUTIONARY PERSPECTIVE 82 (Louis Guillette Jr. & D. Andrew Crain eds., 2000) (setting forth effects of DDT insecticide).

66. See Taisen Iguchi, *Embryonic and Neonatal Exposure to Endocrine-Altering Contaminants: Effects on Mammalian Female Reproduction in* ENDOCRINE DISRUPTING CONTAMINANTS: AN EVOLUTIONARY PERSPECTIVE 234 (Louis Guillette Jr. & D. Andrew Crain eds., 2000) (noting effects when exposed to endocrine disruptors acting like mimics).

67. See SURESH SIKKA ET AL., ENDOCRINE DISRUPTORS AND MALE INFERTILITY, ENDOCRINE DISRUPTORS, 292 (1999) (describing consequences of other endocrine disruptors).

68. See NEWBOLD & JEFFERSON, *supra* note 60, at 48-49 (explaining how some endocrine disruptors prevent normal endocrine function).

69. See HORMONALLY ACTIVE AGENTS, *supra* note 64, at 119 (noting how endocrine could effect wildlife).

70. See *id.* (commenting on specific effects on wildlife).

with test animals.⁷¹ It is, however, still unclear whether endocrine disruptors have the same impact on humans as they do on animals.⁷² Nevertheless, it has been suggested that endocrine disruptors may cause a number of human ailments and reproductive disorders, including but not limited to, the following: “in women, increased cancer rates in the breast, ovary, and uterus . . . in men, increased prostatic and testicular cancer, and poor semen quality associated with subfertility or infertility”⁷³ Given that the endocrine system serves the same regulatory purpose in humans as in animals, it is not difficult to imagine that endocrine disruptors could produce negative effects in humans similar to those produced in animals.

Moreover, some fear that the dangers of endocrine disruptors go well beyond those that may be immediately perceptible. The “fetal origins of adult disease” theory suggests that prenatal exposure to chemicals including endocrine disruptors may not only cause defects readily discernible at birth, such as limb malformations, but may actually be the root of diseases that do not develop until adulthood.⁷⁴ Devra Davis, noted epidemiologist and advisor to the World Health Organization, discusses the possibility that substances like endocrine disruptors may threaten all human life by eliminating males from the species in her book, *WHEN SMOKE RAN LIKE WATER: TALES OF ENVIRONMENTAL DECEPTION AND THE BATTLE AGAINST POLLUTION*.⁷⁵ She points to the facts that more men are having reproductive problems today, fewer boys are being born than just three decades ago, and among the males born, more and more are suffering from genital defects such as undescended testes.⁷⁶ In addition, the relatively low levels of endocrine disruptors found in the environment may not have an observable effect on a healthy adult human, but may still pose a tremendous threat to a fetus developing inside a mother’s womb.⁷⁷

71. See *id.* (explaining similar results reached in lab experiments).

72. See *id.* (noting uncertainty regarding endocrine disruptors effects on humans versus animals).

73. See NEWBOLD & JEFFERSON, *supra* note 60, at 48 (describing specific ailments endocrine disruptors cause in humans).

74. See *id.* at 49-50 (describing “fetal origins of adult disease theory”).

75. See DAVIS, *supra* note 4, at 193 (discussing how endocrine disruptors may eliminate male species).

76. See *id.* (noting issues with male species, including increased reproductive problems).

77. See COLBORN ET AL., *supra* note 5, at 203-04 (commenting on impact of endocrine disruptors on fetuses).

What is disturbing to many people is the seemingly ever-growing presence of endocrine disruptors in the environment and particularly in the water. Thanks in part to improvements in detection methods, endocrine disruptors have been observed in "surface waters (rivers, lakes and marine waters), groundwater, drinking water and sewage treatment effluents."⁷⁸ Residues of antibiotics, hormones, household chemicals, industrial chemicals and numerous pharmaceuticals can be found in water all over the world.⁷⁹ These substances reach the water via a variety of channels including human consumption and excretion, industrial discharge, and disposal of unused materials through sanitary sewer systems.⁸⁰ Many of these materials can pass through wastewater treatment systems unaffected.⁸¹ Another major contributor is stormwater runoff from confined animal feeding operations (CAFOs), where animals are administered high doses of growth hormones and antibiotics.⁸²

Whatever the sources of endocrine disrupting chemicals, their most dramatic consequence may eventually be found in their potentially synergistic qualities. In the late 1990s, a team of researchers at the University of Texas at Austin concluded:

A number of man-made compounds mimic estrogens, although with a lower potency than natural steroidal estrogens. When considered individually, these chemicals may exist in the environment in concentrations too low to be of concern. In combination, however, low dosages of these compounds may act synergistically to produce a strong estrogenic response.⁸³

Previously, experiments on turtles involving polychlorinated biphenyls (PCBs) indicated low-dose synergy of endocrine disrupting

78. See Paul Anderson et al., *Screening Analysis of Human Pharmaceutical Compounds in U.S. Surface Waters*, 38 ENVTL. SCIENCE TECH. 838 (2004) (explaining methods in detection that have lead to increased findings of endocrine disruptors in environment).

79. See *id.* (listing types of endocrine disruptors found in water).

80. See *id.* (describing ways endocrine disruptors reach water).

81. See *id.* (noting how materials can pass through wastewater systems).

82. See Janice M. Skadsen et al., *The Occurrence and Fate of Pharmaceuticals, Personal Care Products and Endocrine Disrupting Compounds in a Municipal Water Use Cycle: A Case Study in the City of Ann Arbor 2*, <http://www.ci.ann-arbor.mi.us/Public-Services/Water/WTP/EndocrineDisruptors.pdf> (Nov. 2004) (commenting on how CAFO's storm water runoff contributes to the problem).

83. See Judith Bergeron et al., *Developmental Synergism of Steroidal Estrogens in Sex Determination*, ENVTL. HEALTH PERSPECTIVES, Vol. 107, No. 2, 1 (Feb. 1999) (describing effect of combinations of low dosage chemicals).

chemicals.⁸⁴ Initially, similar synergy was shown for the pesticides endosulfan and dieldrin; however, the results of these studies could not be duplicated.⁸⁵ The possible synergistic effect of endocrine disruptors is still in dispute and will be resolved only through future scientific research. The potential for several discrete substances to work together to cause endocrine disruption should not be ignored, especially when efforts are made to assess the risks that endocrine disruptors present to human health. A failure to account for the possible additive effects could result in a completely inaccurate risk assessment.

IV. WHAT IS TRADITIONAL ENVIRONMENTAL RISK ASSESSMENT?

A risk assessment can be performed for any activity or substance. Every risk assessment requires judgments to be made by the risk assessor. In numerical terms, risk assessment is the product of the likelihood of an adverse event and the magnitude of the ensuing harm.⁸⁶ For example, assessing the risk of jumping from the top of the Empire State Building can be numerically assessed and then compared to the risk assessment of stepping on a banana peel. The likelihood of an adverse event is very great for a person who jumps from the top of the Empire State Building. It could reasonably be stated as 9.9 out of 10. It is not stated as ten out of ten to allow for a reasonable margin of error. For jumping off the top of the Empire State Building, the magnitude of the ensuing harm, which is certain death, could reasonably be stated as ten out of ten. Therefore, the risk estimate for jumping off the top of the Empire State Building can be numerically represented as ninety-nine, or very risky.

By comparison, if a person were to step on a banana peel, the likelihood of an adverse event is fairly low, despite what cartoons and comedians would have one believe. The likelihood can reasonably be stated as one out of ten. The magnitude of the ensuing harm from stepping on a banana peel is similarly low. Even assuming it actually causes one to slip and fall, it could reasonably be stated as three out of ten. Therefore, the risk estimate for stepping on a banana peel can be numerically represented by three, or not very risky. The two actions can now be compared. Not surprisingly,

84. See *id.* (noting experiments with turtles).

85. See *id.* (finding similar, nonduplicative studies involving pesticides).

86. See Kathy Bunting, *Risk Assessment and Environmental Justice: A Critique of the Current Legal Framework and Suggestions for the Future*, 3 BUFF. ENVTL. L.J. 132 (1995) (noting importance of re-evaluating risk assessment).

jumping from a tall building is assessed as a much more risk-intensive activity than stepping on the skin stripped from a piece of fruit. Although this may be a silly example, it does a good job of demonstrating the fundamentals of risk assessment in a very understandable way.

More specifically, environmental risk assessment is usually described in terms of two components—hazard and exposure.⁸⁷ Hazard is a particular substance's toxicity or ability to cause damage to a living organism.⁸⁸ This is generally determined through laboratory experiments on animals.⁸⁹ Sometimes animal data is supplemented by information about human experience with the particular substance.⁹⁰ In toxicity testing, exposure is the amount or dose of the particular substance being delivered to the target organism in order to assess the degree of harm.⁹¹ Exposure will vary depending on the method of delivery.⁹² Although a substance may be relatively innocuous on the skin, it may be deadly if ingested or inhaled.⁹³

Practically every environmental regulation in the United States utilizes some type of environmental risk assessment.⁹⁴ Every day, environmental risk assessors perform risk assessments to determine the risk of harm to the environment and human health that may result from a particular activity or substance. For example, environmental risk assessors will attempt to determine how much of a particular pollutant may be discharged into a river before there becomes a risk of contamination to fish in that river. Since the mid-1990s, Congress has attempted to include some measure of risk assessment in every new or reauthorized environmental statute.⁹⁵ The belief among the federal legislature is that environmental risk

87. See MARY O'BRIEN, MAKING BETTER ENVIRONMENTAL DECISIONS: AN ALTERNATIVE TO RISK ASSESSMENT 17-25 (2000) (setting forth components of environmental risk assessment).

88. See *id.* (explaining hazard component).

89. See *id.* (describing how to determine hazard).

90. See *id.* (noting combination of human experience with animal studies).

91. See *id.* (explaining method of determining exposure component of environmental risk assessment).

92. See O'Brien, *supra* note 87, at 17-25 (discussing hazard component).

93. See *id.* (noting effect of skin exposure versus ingestion or inhalation).

94. See Bunting, *supra* note 86, at 132 (calling for further examination of link between risk assessment and environmental justice).

95. See Risk Assessment Improvement Act of 1994, H.R. 4306, 103rd Cong. (1994). See also Bunting, *supra* note 86, at 153 (discussing introduction of Risk Assessment Improvement Act of 1994 as congressional bill that would establish risk assessment program in EPA).

assessment will allow regulators to calculate how tax revenue should be used to protect the most human lives per dollar.⁹⁶

Congress has charged the EPA with the duty of performing environmental risk assessments for the federal government. The EPA's general mission is "to protect human health and to safeguard the natural environment—air, water, and land—upon which life depends."⁹⁷ To carry out this mission, the EPA's ORD conducts research in a wide variety of environmental areas in an effort to reduce risks to human health and the environment based upon the best available scientific information.⁹⁸ A major component of the ORD is the National Center for Environmental Assessment (NCEA).⁹⁹ The NCEA is the primary environmental risk assessment agency in the country.¹⁰⁰ The NCEA conducts environmental risk assessments, develops ways to improve them, and provides guidance to other environmental risk assessors.¹⁰¹

The NCEA's three major work areas include conducting: (1) environmental risk assessment; (2) methods research; and (3) environmental risk assessment guidance.¹⁰² It conducts environmental risk assessments for substances of national significance such as dioxin, mercury and trichloroethylene.¹⁰³ The NCEA conducts methods research to improve the "state-of-the-science" of environmental risk assessment by developing new and scientifically defensible environmental risk assessment methods based upon the latest advances in science and technology.¹⁰⁴ Finally, the NCEA provides support to outside risk assessors through consultations, training, and scientific information such as the dioxin emissions inventory.¹⁰⁵ According to the NCEA, environmental risk assessment is:

96. See Bunting, *supra* note 86, at 170 (outlining principles on risk assessment drawn up by sub-committee of Office of Management and Budget).

97. See U.S. Env'tl. Protection Agency: History, <http://www.epa.gov/history/> (last visited Apr. 19, 2006) (providing background of EPA).

98. See U.S. Env'tl. Protection Agency: Research & Development, <http://www.epa.gov/ord/htm/aboutord/htm> (stating how ORD carries out EPA's mission).

99. See U.S. Env'tl. Protection Agency: National Center for Environmental Assessment, <http://cfpub.epa.gov/ncea/cfm/aboutncea.cfm?ActType=aboutNCEA> (noting NCEA contribution to ORD).

100. See *id.* (noting NCEA's primary role).

101. See U.S. Env'tl. Protection Agency: Strategic Plan for the Office of Research and Development 62 (May 1996), <http://www.epa.gov/osp/strtpplan/documents/ord96strplan.pdf> [hereinafter Strategic Plan] (outlining NCEA's modus operandi).

102. See *id.* (listing NCEA's three major work areas).

103. See *id.* (noting chemicals on which NCEA conducts risk assessments).

104. See *id.* (stating goals of NCEA's methods of research).

105. See *id.* (noting how NCEA supports outside risk assessors).

[T]he probability that a harmful consequence will occur as a result of an action. Risk is a function of hazard and exposure. For risk to occur, there must be a source of risk (hazard) and an exposure to the hazard. Risk assessment is the process by which one attempts to evaluate and predict the likelihood and extent of harm that may result from a health or environmental hazard. Risk assessment provides essential information about the severity and extent of specific environmental problems for use in EPA risk management decisions.¹⁰⁶

When the EPA performs what it calls “hazardous pollutant risk assessments,” it uses benchmark dose (BMD) methods.¹⁰⁷ The BMD methods work by estimating reference doses (RfDs) and reference concentrations (RfCs) which are used with other scientific information to establish standards for non-cancer human health effects.¹⁰⁸ In the past, RfDs and RfCs were determined on the basis of no-observed-adverse-effect levels (NOAELs).¹⁰⁹ The NOAELs are simply the highest dose administered in an animal experiment where there was no documented adverse health effect.¹¹⁰ The BMD methods involve applying mathematical models to dose-response data and using the results to formulate benchmark responses (BMR) such as a certain percentage increase in the incidence of a particular size and type of tumor as a result of a certain dose of a particular contaminant.¹¹¹

To facilitate the use of the BMD method, the EPA has developed Benchmark Dose Software (BMDS).¹¹² Utilizing the BMDS is a four-step process. First, a data set is created using the BMDS spreadsheet capability.¹¹³ An existing data set can also be imported from another source such as a Lotus spreadsheet file.¹¹⁴ Second, an appropriate model must be selected based upon the nature of the data being evaluated through the BMDS.¹¹⁵ Third, the run pa-

106. See Strategic Plan, *supra* note 101, at <http://www.epa.gov/osp/strtpplan/documents/ord96strplan.pdf> (defining environmental risk assessment).

107. See Benchmark Dose Software, 65 Fed. Reg. 35,925 (June 6, 2000) (stating particular method used in hazardous pollutant risk assessment).

108. See *id.* (describing how BMD methods work).

109. See *id.* (explaining how RfDs and RfCs were determined in past).

110. See *id.* (defining NOAELs).

111. See *id.* (explaining how BMD methods work).

112. See Benchmark Dose Software, 65 Fed. Reg. 35,925 (June 6, 2000) (noting software developed to facilitate use of BMD method).

113. See *id.* (stating step one of utilizing BMDS).

114. See *id.* (noting another method of getting data sets).

115. See *id.* (stating second step in utilizing BMDS).

rameters and run options of the selected model must be specified.¹¹⁶ Fourth, the selected model is run and textual and graphical results can then be viewed.¹¹⁷ The BMDS can be downloaded from the EPA website, and the EPA encourages professional environmental risk assessors to use the BMDS and to submit feedback including criticism of the software.¹¹⁸

To establish a regulatory scheme, the EPA must first determine those pollutants that it believes pose the greatest risk of harm to human health.¹¹⁹ Then, the EPA determines what treatments are available or could be developed to manage those risks and what the associated costs would be for a particular treatment strategy.¹²⁰ The EPA does not, however, make these determinations without assistance.¹²¹ The NCEA plays a major role in the EPA's environmental or ecological risk assessment process. The NCEA performs the actual risk assessment research to characterize the adverse health effects that result from exposure to pathogenic or toxic agents.¹²² First, the NCEA compiles scientific data regarding human health and exposure that will be utilized in analytical models.¹²³ Next, the NCEA develops analytical models for qualitative and quantitative estimates of risks associated with various pollutants.¹²⁴ Finally, the NCEA interprets the output of the analytical models to predict potential risks to human health from various contaminants.¹²⁵

Currently, the EPA and the NCEA are working to develop a comprehensive approach for various chemicals and microbes in drinking water. The focus is on devising a comparative risk model to measure risks resulting from exposure to multiple pollutants at play in a single drinking water source. Exposure to multiple pollutants may result in a variety of health hazards such as gastrointestinal illness, cancer or even reproductive disorders. This kind of multiple risk analysis is the most difficult to perform, but it is ex-

116. *See id.* (stating third step in utilizing BMDS).

117. *See* 65 Fed. Reg. 35,925 (stating fourth step in utilizing BMDS).

118. *See id.* (noting where one can download BMDS).

119. *See* Peer Review of EPA Draft Human Health and Ecological Risk Assessment of Perchlorate, 67 Fed. Reg. 75 (Jan. 2, 2002) (stating first step in establishing regulatory schemes); Notice of Public Information Collection Being Reviewed by the Federal Communications Commission, 67 Fed. Reg. 76 (Jan. 2, 2002).

120. *See id.* (noting EPA procedures related to establishing regulatory scheme).

121. *See id.* (noting NCEA role in establishing a regulatory scheme).

122. *See id.* (stating NCEA procedures).

123. *See id.* (discussing NCEA's first step regarding data compilation).

124. *See* Peer Review of EPA Draft Human Health and Ecological Risk Assessment of Perchlorate, 67 Fed. Reg. 75 (noting second step of model development).

125. *See id.* (noting final NCEA step).

actly the kind that may be necessary for assessing the human health risk of endocrine disruptors if it is eventually proved that they have a synergistic effect in drinking water. As challenging as this task is, it is not the first time the EPA has encountered it. In fact, the EPA has developed guidelines for assessing the health risks of chemical mixtures.¹²⁶

V. HOW IS WATER REGULATED?

The Federal Water Pollution Control Act (FWPCA),¹²⁷ more commonly known as the Clean Water Act (CWA),¹²⁸ regulates the discharge of all pollutants into the navigable waters of the United States.¹²⁹ The CWA requires limits be set for discharges of effluent based upon water quality standards.¹³⁰ Individual states establish actual water quality standards.¹³¹ Water quality standards must include the designated use or uses for the waters in question and the criteria necessary for protecting the designated use or uses.¹³² When an individual state adopts a water quality standard, the EPA must review and either approve or disapprove it.¹³³ To assist the states in creating water quality standards, the EPA publishes nationally recommended water quality criteria based upon the latest available scientific knowledge.¹³⁴ These criteria provide the states with guidance for determining water quality standards, but they are not regulations themselves.¹³⁵

One of the nationally recommended water quality criteria published by the EPA is the Human Health Criteria Calculation Matrix.¹³⁶ Human health criteria for water quality are numeric values

126. See Guidelines for the Health Risk Assessment of Chemical Mixtures, 51 Fed. Reg. 34,014 (Sept. 24, 1986) (discussing EPA guidelines).

127. 33 U.S.C. §§ 1251-74 (2000).

128. Clean Water Act, 33 U.S.C. § 1314 (2000).

129. See Water Pollution Control Act, 33 U.S.C. §§ 1251-74 (2000) (noting discharge regulations).

130. See 33 U.S.C. § 1314(b)(1)(B) (listing factors considered for measures and practices to be applicable to point sources).

131. See Water Quality Standards General Provisions, 40 C.F.R. pt. 131.4(a) (2006) (noting state responsibility).

132. See *id.* pt. 131.2 (discussing purpose of water quality standards).

133. See *id.* pt. 131.5(a) (stating EPA review guidelines).

134. See 33 U.S.C. § 1314(a) (noting development of and publication of criteria).

135. See *id.* § 1314(a)(7) (noting guidance to states).

136. See U.S. Env'tl. Protection Agency: Human Health Criteria Calculation Matrix (Nov. 2002), http://www.epa.gov/ost/criteria/hh_calc_matrix.pdf [hereinafter Human Health Criteria Calculation Matrix] (discussing information relating to calculation of human health criteria).

for allowing a certain level of a pollutant while still being protective of human health.¹³⁷ Criteria are based upon risk assessments derived from toxicity studies on laboratory animals and human data when available.¹³⁸ Currently, some endocrine disruptors such as PCBs can be found in the Human Health Criteria Calculation Matrix, but they are not in the matrix as a result of endocrine disrupting qualities.¹³⁹ They are there because of their carcinogenic attributes.¹⁴⁰ At the present time, there are no regulations based upon the CWA that target endocrine disruptors for their endocrine disrupting properties, but that is not to say they could not be developed at some point in the future. It is much more likely, however, that regulation of endocrine disruptors will come as a result of the SDWA rather than CWA because the SDWA has been amended to specifically address endocrine disruptors, at least with regard to screening and testing.¹⁴¹

The SDWA requires the EPA to establish standards for drinking water.¹⁴² Consequently, the EPA creates Maximum Contaminant Levels Goals (MCLGs) for drinking water.¹⁴³ Individual contaminants are evaluated for possible human health effects, and MCLGs are created with a reasonable margin of safety.¹⁴⁴ These goals are not legally enforceable and may not even be achievable with currently available drinking water treatment technology.¹⁴⁵ For example, MCLGs are often set at zero. As a result, the EPA also creates Maximum Contaminant Levels (MCLs) based upon MCLGs.¹⁴⁶ When EPA establishes MCLs, it considers costs and ensures the levels are actually feasible with currently available drinking water treatment technology.¹⁴⁷ Water that comes out of a tap must satisfy MCLs. MCLs are legally enforceable standards.¹⁴⁸

137. See *id.* (explaining human health criteria).

138. See U.S. Env'tl. Protection Agency: National Recommended Water Quality Criteria (2002), <http://www.epa.gov/waterscience/criteria/nrwqc-2002.pdf> (noting what criteria are based on). Current and other past National Recommended Water Quality Criteria can be found at <http://www.epa.gov/waterscience/criteria/wqcriteria.html>.

139. See Human Health Criteria Calculation Matrix, *supra* note 136 (noting evidence of PCBs).

140. See *id.* (noting reason for PCBs in Matrix).

141. See 42 U.S.C. § 300j-17 (2000) (noting SDWA screening program).

142. See *id.* §§ 300f to 300j-26 (setting standards).

143. See *id.* § 300g-1 (stating goals).

144. See *id.* (explaining evolution process for contaminants).

145. See *id.* (stating limitations on goals).

146. See 42 U.S.C. § 300g-1 (2000) (establishing MCL creation).

147. See *id.* (setting forth process of MCL creation).

148. See *id.* (noting MCL legal status).

Both MCLGs and MCLs are based upon human health effects as determined by various risk assessments.¹⁴⁹ In conducting such risk assessments, the EPA is required under the SDWA to use “the best available, peer-reviewed science and supporting studies conducted in accordance with sound and objective scientific practices; and data collected by accepted methods or best available methods”¹⁵⁰ The SDWA also requires that when the EPA creates a regulation based upon a risk assessment, it must identify the following:

- (i) each population addressed by any estimate of public health effects;
- (ii) the expected risk or central estimate of risk for the specific populations;
- (iii) each appropriate upper-bound or lower-bound estimate of risk;
- (iv) each significant uncertainty identified in the process of the assessment of public health effects and studies that would assist in resolving the uncertainty; and
- (v) peer-reviewed studies known to the [EPA] Administrator that support, are directly relevant to, or fail to support any estimate of public health effects and the methodology used to reconcile inconsistencies in the scientific data.¹⁵¹

There is no question that if endocrine disrupting compounds are eventually regulated under the SDWA, such regulation would be based upon human health effects as determined by risk assessments.

As with most current federal regulations, the regulation of drinking water tends to be developed through a process. The EPA is not yet at the point in the process where it is conducting risk assessments for endocrine disruptors.¹⁵² Presently, the EPA is still trying to develop screening and testing methods for endocrine disruptors.¹⁵³ There are an estimated 87,000 chemicals that should be

149. *See id.* § 300(g)(b)(3)(A) (stating risk assessment process).

150. *See id.* (noting which studies and data are to be used).

151. *See* 42 U.S.C. § 300(g)(b)(3)(B) (stating elements of proper regulation promulgation).

152. *See* U.S. ENVTL. PROTECTION AGENCY, REPORT TO U.S. HOUSE OF REPRESENTATIVES COMMITTEE ON APPROPRIATIONS ON THE STATUS OF THE ENDOCRINE DISRUPTOR METHODS VALIDATION SUBCOMMITTEE 6 (May 2002) (informing committee on status of risk assessment process).

153. *See id.* (noting current position of EPA).

screened for the potential to cause endocrine disruption.¹⁵⁴ Based upon the EDSTAC recommendations, the EPA has focused its initial screening and testing on compounds that affect estrogen, androgen and thyroid (EAT) hormones because they are important with regard to reproduction, development and growth in both humans and wildlife.¹⁵⁵

The EPA's basic approach is comprised of three components: (1) Priority Setting; (2) Tier One Screening; and (3) Tier Two Testing.¹⁵⁶ The EPA's Priority Setting focuses on which potential endocrine disruptors have the most potential for exposure to humans.¹⁵⁷ The Tier One Screening will develop screening assays to identify compounds that have the potential to interact with the EAT hormones.¹⁵⁸ The Tier Two Testing program has the following three goals:

- (1) Determining whether a substance may cause endocrine-mediated effects through or involving estrogen, androgen, or thyroid hormone systems,
- (2) Determining the consequences to the organism of the activities observed in Tier 1, and
- (3) Establishing the relationship between doses of an endocrine-active substance administered in the test and the effects observed.¹⁵⁹

Once data have been collected from Tier One Screening and Tier Two Testing, the EPA will turn its attention to characterizing the potential risk presented by endocrine disruptors.¹⁶⁰

The EPA intends to perform an "exposure assessment" to characterize human exposure to endocrine disruptors.¹⁶¹ The EPA will also perform a "hazard assessment" for endocrine disruptors by collecting the hazard data resulting from the Tier One Screening and the Tier Two Testing stages and then comparing it to other relevant

154. See ENDOCRINE DISRUPTOR SCREENING AND TESTING ADVISORY COMMITTEE, EDSTAC FINAL REPORT, EXECUTIVE SUMMARY, ES-3 (1998) (estimating number of chemicals recommended for testing).

155. See *id.* (concluding which compounds should be tested first).

156. See Endocrine Disruptor Screening Program; Proposed Statement of Policy, 63 Fed. Reg. 71,542 (Dec. 28, 1998) (setting forth EPA testing process).

157. See *id.* (noting importance of protecting human health).

158. See *id.* (identifying goals of Tier One screening).

159. See U.S. ENVIRONMENTAL PROTECTION AGENCY, ENDOCRINE DISRUPTOR SCREENING PROGRAM—EDSP COMPONENTS, <http://www.epa.gov/scipoly/oscpendo/edspoverview/components.htm> (last visited Apr. 19, 2006) [hereinafter ENDOCRINE DISRUPTOR SCREENING] (stating goals of Tier Two Testing).

160. See *id.* (explaining EPA characterization process).

161. See *id.* (identifying exposure assessment process).

hazard information.¹⁶² Finally, the EPA will conduct the actual "risk assessment" for endocrine disruptors by integrating the information from the exposure assessment and the hazard assessment.¹⁶³ Basically, the EPA will look at the amount of exposure humans have to endocrine disruptors to determine the potential for harm from such a level of exposure. It appears that the EPA only intends to look at the independent effects of individual endocrine disruptors or classes of endocrine disruptors rather than undertaking the even more complex task of considering the possibly interactive (e.g., synergistic) effects of endocrine disruptors.

The EPA's immediate focus with regard to endocrine disruptors is implementation of the EDSP.¹⁶⁴ As a result, the EPA has assigned staff from its Office of Water, Office of Pesticide Programs, Office of Pollution Prevention and Toxics, Office of Science Coordination and Policy and Office of General Counsel to participate in an endocrine disruptors Regulatory Activities Workgroup.¹⁶⁵ The workgroup meets once a week to address issues as they arise from the implementation of the EDSP.¹⁶⁶ Presently, the workgroup is focusing on questions surrounding data collection, scope of the EDSP and sources of drinking water.¹⁶⁷ This work is complicated and time intensive. As of February 2004, the workgroup was still reviewing comments submitted in response to the EDSP notice published in the Federal Register in 1998.¹⁶⁸

Currently, there is no regulation of endocrine disrupting chemicals in water in the United States. Although the CWA does regulate all discharges of pollutants to the navigable waters of the United States, the first regulation of endocrine disrupting chemicals with regard to water could possibly result from the SDWA. Because the SDWA already has a provision requiring the EPA to develop a screening and testing program for endocrine disruptors, it appears that the initial regulation of endocrine disruptors may be in drinking water. At present, the EPA is still struggling with the issues related to the development and implementation of the

162. *See id.* (creating hazard assessment process).

163. *See id.* (explaining final risk assessment creation).

164. *See* ENDOCRINE DISRUPTOR SCREENING, *supra* note 159 (noting main goals of EDSP implementation).

165. *See id.* (listing various participants in EDSP process).

166. *See id.* (explaining function of EDSP staff).

167. *See id.* (stating workgroup also analyzes confidential business information, cost-sharing provisions, exemptions, statutory authorities and substantial population).

168. *See id.* (noting workgroup's continuing efforts to review Federal Register comments to EDSP).

screening and testing program for endocrine disruptors. Once those issues are satisfactorily addressed, the result will be screening and testing data that will be used for conducting risk assessments. The results of such risk assessments will be used by policy makers as the basis for determining how best to regulate endocrine disrupting compounds. This is the same process that has historically been used by the EPA to develop regulations for numerous drinking water pollutants in the past.

VI. HOW WAS RADON IN DRINKING WATER REGULATED?

Radon is an odorless, colorless and radioactive noble gas.¹⁶⁹ Therefore, a person cannot detect radon with any of the senses. Radon results from the natural radioactive decay of radium, which is a byproduct of uranium in the earth's crust.¹⁷⁰ In addition, radon decay forms solid isotopes, which are also undetectable by human senses and are readily transported when dissolved in water.¹⁷¹ Most radon forms from decaying rocks in the top thirty feet of soil.¹⁷² Underground radon is often carried to the surface by groundwater where it is released into the air.¹⁷³ Breathing in radon is second only to cigarette smoking as a cause of lung cancer in the United States.¹⁷⁴ Ingestion of radon in drinking water is also known to cause cancer in other organs including the stomach.¹⁷⁵ Therefore, radon in water poses risks from both ingestion and inhalation.¹⁷⁶ When using water contaminated with radon to shower or for other household purposes, the radon can be released and

169. See U.S. ENVIRONMENTAL PROTECTION AGENCY, A CITIZEN'S GUIDE TO RADON: THE GUIDE TO PROTECTING YOURSELF AND YOUR FAMILY FROM RADON, 402-K-02-66, at 3 (Sept. 2005), <http://www.epa.gov/radon/images/citizensguide.pdf> [hereinafter CITIZEN'S GUIDE TO RADON] (explaining characteristics of radon).

170. See *id.* at 4 (describing formation of radon).

171. See J.R. Garcia-Vindas & M.M. Monnin, *Radon Concentration Measurements in the Presence of Water and its Consequences for Earth Science Studies*, 39 RADIATION MEASUREMENTS 3, 319-22 (June 2004) (explaining consequences of solid isotopes, which are formed by radon decay).

172. See *id.* (noting typical formation of radon is from decaying rocks).

173. See L. Villalba et al., *Radon Concentrations in Ground and Drinking Water in the State of Chihuahua, Mexico*, 80 J. ENVTL. RADIOACTIVITY 2, 139-51 (Dec. 2004) (explaining high levels of radon attributed to nature of aquifer rocks).

174. See CITIZEN'S GUIDE TO RADON, *supra* note 169, at 3 (explaining warning by Surgeon General).

175. See *id.* at 8 (stating research has shown risk of lung cancer from breathing radon in air is much larger than risk of stomach cancer from swallowing water with radon in it).

176. See *id.* (explaining risks of radon in water).

inhaled.¹⁷⁷ Once these risks were medically confirmed, there was a call for federal regulation of radon in drinking water.¹⁷⁸

The 1986 Amendments to the SDWA required the EPA to set a standard for radon in drinking water regulating it along with several other radionuclides.¹⁷⁹ Due to delays in the creation of these regulations, the EPA was sued and eventually agreed to promulgate regulations by 1993.¹⁸⁰ Because radon was known to cause cancer, its MCLG was set at zero.¹⁸¹ After consideration of costs, feasibility and the need to protect human health, the EPA proposed a radon MCL of 11,000 becquerel per cubic meter.¹⁸² The majority of public comment on the proposed MCL was critical of the relatively low limit and suggested a higher level for radon.¹⁸³ In 1992, Congress intervened and directed the Office of Technology Assessment to review the EPA's radon analysis.¹⁸⁴ Around the same time, the Office of Management and Budget took issue with the EPA's projected mitigation costs for radon.¹⁸⁵ Consequently, in 1994, Congress ordered the EPA to hold off promulgating a final standard for radon in drinking water.¹⁸⁶

By way of the 1996 Amendments to the SDWA, Congress required the EPA to perform a risk assessment for radon as a precursor to new drinking water regulation.¹⁸⁷ The SDWA states in relevant part:

Prior to proposing a national primary drinking water regulation for radon, the [EPA] Administrator shall arrange for the National Academy of Sciences to prepare a risk assessment for radon in drinking water using the best availa-

177. See *id.* (explaining most of risk from radon in water comes from radon released into air when water is used for showering and other household purposes).

178. See Philip Shabecoff, *Measures Offered in Congress on Detection and Cleanup of Radon*, N.Y. TIMES, Mar. 13, 1987, at A15 (stating members of Congress introduced legislation for federal program to help detect and deal with radon).

179. See NAT'L RESEARCH COUNCIL, *RISK ASSESSMENT OF RADON IN DRINKING WATER*, 17 (1999) (noting standards and regulations for radon in drinking water created by 1986 amendments to SDWA).

180. See *id.* at 9 (stating EPA was sued over delays in effectuating regulations).

181. See *id.* (stating MCLG of radon).

182. See *id.* (explaining factors considered to formulate radon MCL).

183. See *id.* at 10 (noting criticism of public comment on proposed MCL).

184. See NAT'L RESEARCH COUNCIL, *supra* note 179, at 10 (stating Congress intervened in order to address and assess radon analysis).

185. See *id.* (noting controversy regarding EPA's projected mitigation costs of radon).

186. See 42 U.S.C. § 300g-13(B)(i) (2000) (explaining Congress's response to criticism of handling radon in drinking water).

187. See Safe Drinking Water Act, 42 U.S.C. § 300j-17 (1994 & Supp. 111 1997) (citing where EPA is given power to screen for contaminants).

ble science The risk assessment shall consider each of the risks associated with exposure to radon from drinking water and consider studies on the health effects of radon at levels and under conditions likely to be expected through residential exposure.¹⁸⁸

The statute also required peer review of the risk assessment for radon in drinking water.¹⁸⁹ The SDWA gave the EPA thirty months to prepare a risk assessment for radon in drinking water including a cost analysis for a potential radon MCL.¹⁹⁰ Once the risk assessment was completed and made available for public comment, the EPA would have six months to actually propose a MCL for radon.¹⁹¹ After the proposal of the radon MCL, the EPA would have one year to promulgate a final radon MCL and related national primary drinking water regulations.¹⁹²

The 1996 Amendments to the SDWA also provided for an Alternative Maximum Contaminant Level (AMCL) for radon.¹⁹³ If it was determined that the proposed MCL for radon was “more stringent than necessary to reduce the contribution to radon in indoor air from drinking water to a concentration that is equivalent to the national average concentration of radon in outdoor air,” the EPA would be required to promulgate an AMCL for radon.¹⁹⁴ In order to comply with the AMCL, states would need to develop multimedia radon mitigation programs including “public education, testing, training, technical assistance, remediation grant and loan or incentive programs, or other regulatory or nonregulatory measures.”¹⁹⁵ The EPA had to approve a state’s multimedia mitigation program, which would be reviewed every five years to ensure that human health benefits of complying with the AMCL were equal to or better than compliance with the MCL.¹⁹⁶

188. See 42 U.S.C. § 300g-13(B)(i) (quoting SDWA risk assessment requirements).

189. See *id.* § 300g-13(B)(iii) (noting regulation mandated peer-revision for risk assessments).

190. See *id.* § 300g-13(C) (discussing requirements of regulation).

191. See *id.* § 300g-1(b)(13)(D) (discussing timeframe of proposed regulation).

192. See *id.* § 300g-1(b)(13)(E) (describing final radon regulation process).

193. See 42 U.S.C. § 300g-1(b)(13)(F) (explaining maximum contaminant levels).

194. See *id.* (discussing alternative maximum contaminant levels).

195. See *id.* § 300g-1(b)(13)(G)(ii) (describing elements of multimedia radon mitigation programs).

196. See *id.* § 300g-1(b)(13)(G)(iii) (explaining approval of radon mitigation programs).

Once the EPA was ready to issue a final MCL and AMCL for radon, it also was required to promulgate guidance or regulations "describing the best treatment technologies, treatment techniques, or other means" for reducing the level of radon in drinking water.¹⁹⁷ In assessing treatment technology, the EPA must consult with the states and drinking water providers to consider the availability, costs and effectiveness of the technology both in the laboratory and the field.¹⁹⁸ The SDWA also required the EPA to consider the quality of source water when evaluating treatment technology.¹⁹⁹ Finally, in completing its evaluation of available treatment techniques for radon reduction or removal, the EPA had to disclose any assumptions it made with regard to public health including human health risk assessments.²⁰⁰

Conducting the human health risk assessment for radon in drinking water was not an easy task. Data on radon in water varied greatly across the country.²⁰¹ The Rocky Mountain states, the Appalachian states and the New England states all had public water supplies with elevated levels of radon in comparison to the rest of the nation.²⁰² The amount of radon in water that is released into air is calculated by the "transfer coefficient," which is estimated by a mathematical model.²⁰³ The biological effect of radon exposure is believed to be the result of single alpha particles damaging DNA and leading to genetic instability as evidenced by tumor growth.²⁰⁴ There are no studies that quantify the cancer risk from ingestion of water contaminated with radon, so the risk is determined by mathematical models that calculate human tissue's ability to absorb radon.²⁰⁵ A physiologically-based pharmacokinetic (PBPK) model was developed for radon.²⁰⁶ The PBPK model calculates the behavior of radon in the human body by simulating the ingestion, rate of

197. See *id.* § 300g-1(b)(15) (describing variance technologies).

198. See 42 U.S.C. § 300g-1(b)(15)(A) (2000) (discussing Administrator's duties when promulgating water regulations).

199. See *id.* § 300g-1(b)(15)(B) (describing limitations of variance technologies).

200. See *id.* § 300g-1(b)(15)(C) (indicating requirement to disclose assumptions in regulations identifying variance technologies).

201. See NAT'L RESEARCH COUNCIL, *supra* note 179, at 12 (discussing data on radon).

202. See *id.* (pointing out inconsistent radon levels throughout country).

203. See *id.* (indicating method for predicting radon's rate of release).

204. See *id.* (discussing genetic effects of radon pursuant to exposure).

205. See *id.* at 14 (analyzing radon's risks).

206. See NAT'L RESEARCH COUNCIL, *supra* note 179, at 14 (identifying model used to examine radon).

dissolution into blood, transfer through the bloodstream and ultimate deposition and retention in human tissue.²⁰⁷

A number of mathematical models were used to generate risk estimates for ingestion and inhalation of radon via drinking water.²⁰⁸ These models utilized factors such as amount of water ingested, duration of exposure and rate of water to air transfer.²⁰⁹ The models relied on numerous assumptions. For example, it was assumed that "all the radon remained dissolved in the water during the transfer process" to air.²¹⁰ The risk estimates for ingestion were then compared to annual cancer deaths in the United States.²¹¹ The EPA conducted a similar analysis for inhalation, and it was determined that the greatest risk of cancer from radon in drinking water results from emission into air and subsequent inhalation and not from direct ingestion.²¹² The EPA determined that "inhalation accounts for about 89% of the estimated cancer risk and ingestion accounts for 11%."²¹³

The EPA fully acknowledged that its risk assessment for radon in drinking water was based upon an analysis that contained a certain level of uncertainty. In fact, the related risk assessment document produced by the National Academy of Sciences for the EPA explicitly states:

Estimating potential human exposures to and health effects of radon in drinking water involves the use of large amounts of data and the use of models for projecting relationships outside the range of observed data. The data and models must be used to characterize population behaviors, engineered-system performance, contaminant transport, human contact, and dose-response relationships among populations in different areas, so large variability and uncertainties are associated with the resulting risk characterization.²¹⁴

207. *See id.* (examining method of simulating radon's behavior in human body).

208. *See id.* at 15 (discussing models used to obtain risk estimates for radon).

209. *See id.* at 17-18 (describing factors used in radon models).

210. *See id.* at 18 (illustrating necessary assumptions models relied upon).

211. *See* NAT'L RESEARCH COUNCIL, *supra* note 179, at 16 (analyzing ingestion risk estimates compared to cancer deaths).

212. *See id.* (discussing origins of cancer risks from radon).

213. *See id.* at 17 (breaking down sources into component percentages).

214. *See id.* at 18 (describing data and models involved estimating radon effects).

The reality is that all risk assessments rely to some degree on models built on assumptions and judgments of risk assessors. The resultant conclusions are estimations at best, but the fact that they are built on assumptions and judgments does not completely invalidate the use of risk assessments. The possibility for uncertainty inherent in the risk assessment process demands the use of the best available data. For example, because there was data that suggested the real risk of cancer from radon in drinking water came from inhalation rather than ingestion, the EPA closely scrutinized and incorporated those data into the risk assessment. As a result, EPA proposed an AMCL that allowed for multimedia mitigation rather than just a traditional regulatory MCL for radon. Similarly, because there are data suggesting endocrine disrupting compounds may have additive or synergistic effects, those data should also be fully considered. Such considerations complicate the regulatory process. Due to the complications in the risk assessment for radon, the EPA still does not have a standard for radon in drinking water and is not expected to have one anytime soon.

VII. WHY WILL THIS APPROACH FAIL FOR EDCs?

There is no question that there are synthetic chemicals that mimic naturally occurring hormones and that these chemicals may produce physiological responses similar to those produced naturally, but there are still a lot of unanswered questions surrounding endocrine disrupting compounds. One of the biggest questions about endocrine disruptors is whether they can act synergistically. While there is a strong body of work showing that certain estrogenic endocrine disruptors cause reproductive effects in certain species of fish at environmentally relevant levels, there is relatively little evidence that environmental exposure to endocrine disrupting chemicals can cause adverse effects in humans. Moreover, the concentration levels of endocrine disruptors in the environment are simply not well-characterized at the present time.

It would seem that the obvious solution is to test individual chemicals to see what endocrine effect they have, then test combinations of endocrine disruptors for their effects and, finally, measure the levels of endocrine disruptors currently in the environment. At this time, however, scientists are not even certain which tests would be good indicators of physiological consequences; the EPA currently is struggling to develop test methods. To some degree, the media, the public and non-governmental organizations are pushing the EPA to develop tests. Endocrine dis-

ruptors can be a very volatile subject. Presently, scientists are trying to generate data sets to show dose-responses between herbicides and frog deformations, but the public may demand immediate regulation of endocrine disruptors in drinking water. Any such regulation would need to be based upon risk assessments. The models needed for such risk assessments have certainly not yet been developed, especially models that could account for endocrine disruptors' possibly synergistic effects.

From a regulatory standpoint, endocrine disruptors are an extremely difficult issue because there are many different sources of these compounds. For example, personal care products are reaching drinking water sources such as rivers via sewer systems. Ultimately, the only way to regulate some of the synthetic chemicals may be to eliminate them from commerce, but there is a long way to go before that decision could be made. The regulation of endocrine disruptors in drinking water should probably start with a monitoring program. An agency could collect water samples and measure concentration levels under different conditions. These data would then be compared against a known toxicity standard. Setting a water quality standard for endocrine disruptors, however, will not be easy because it leads back to the problems with developing accurate methodologies for screening and testing.

The issue of endocrine disruptors is not going to be resolved quickly, and it may draw resources away from other areas where there could be greater impact in terms of protecting water quality. Programs such as buying open space along water bodies to create buffer zones, for example, is one area where spending dollars might be more beneficial. Endocrine disruptors could get in the way of making significant advancement in other more pressing environmental issues for they may not even present a significant health risk, particularly when compared to other environmental problems.

The human health issues related to endocrine disruptors are very problematic. To begin, the public has an unrealistic desire to have "pure water" coming out of the tap. People do not want to be told that their drinking water has minute traces of estrogen. The reality, however, is that technology is not to the point that there can be complete removal of such substances. In fact, the costs of treatment to reach just non-detect levels can be very high for any contaminant. Also, the multiple types of endocrine disruptors may require different treatment technologies to remove them, and each treatment technology has its own costs. The total costs of removing every possible endocrine disrupting compound could quickly be-

come astronomical. Although the public may want pure water, people are not prepared to pay what it would actually cost even if sufficient technology did exist.

Furthermore, there are many barriers to regulating endocrine disruptors. By far, the biggest barrier is a cause and effect issue. The body's endocrine system reacts to chemicals all the time, and it is very challenging to determine at what point a chemical is causing a significant and negative change in the endocrine system. It is especially difficult to link a specific endocrine disruptor to an adverse impact on the human body because there are so many other possible factors at play. To date, no definitive link has been established between human health risks and environmental exposure to endocrine disruptors. There are two reasons for the lack of a definitive link: first, there are few human studies on the effects of endocrine disruptors, and available epidemiological studies are inconclusive due to environmental contaminants. Second, it is difficult to extrapolate from lab animals to human health because many co-occurring endocrine disruptors complicate human and lab animal studies.

The EPA's Endocrine Disruptor Screening Program is still in a very early phase. The EPA is trying to establish accurate methods for measuring concentrations and effects of endocrine disruptors. The ultimate goal could be for the EPA to set standards for drinking water facilities. However, the irony is that under TSCA and FIFRA, the federal government is currently allowing these substances to be used and discharged into the environment. There is still tremendous uncertainty about the approval process for pharmaceuticals and chemicals. It is not clear whether the current screening process for bringing new pharmaceuticals and other chemicals to market is adequate to address the eventual impact on the environment or drinking water. Again, there is a lack of cause and effect evidence. There is a real question as to the proper way to regulate. It might be more feasible to ban the use of an endocrine disruptor or otherwise prevent it from reaching source water (e.g., source water protection programs) rather than try to remove it from drinking water.

Another area of growing concern is animals or agriculture as sources of endocrine disruptors. Farm waste and pet waste could be an enormous source of endocrine disruptors to water bodies via urban and suburban storm water runoff. Like so much in the field of endocrine disruptors, there is just not enough information yet to make any kind of an informed decision. More information is

needed to link cause and effect. A great deal more treatment or removal research is needed, especially to make removal of endocrine disruptors feasible, let alone cost effective. Moreover, current regulation should be made more consistent before new legislation is passed. The EPA should become a bigger part of the approval process for new chemicals and pharmaceuticals, rather than being asked to deal with an approved substance after it has been discharged into the environment.

For the federal government to regulate endocrine disrupting compounds as drinking water pollutants, it must eventually conduct risk assessments. If the EPA uses traditional environmental risk assessments for endocrine disruptors, it will be looking at the individual effects of separate potentially endocrine disrupting compounds. Relying on the results of such studies, the EPA could develop mathematical risk assessment models that would eventually lead to MCLs for individual endocrine disruptors. However, because there is some evidence to suggest that certain compounds at low levels act together to cause endocrine disruption, such MCLs might be inadequate because they would still allow various compounds to be discharged into the environment at low levels where they would be capable of acting in combination with other compounds and still cause endocrine disruption.

If the issue of regulating endocrine disruptors as drinking water pollutants based upon traditional environmental risk assessment is not examined now, it is likely that the regulatory process will continue as usual. The result could be federal regulations that are devastatingly expensive for drinking water utilities, without significant public health benefit, or huge federal spending to create regulations that could still be challenged by the regulated community and ultimately invalidated by the courts. Either way, trillions of dollars could be spent with little real benefit for water quality or human health unless the issue is addressed today. The only way to address this issue is to encourage and support endocrine disruptor research, especially scientific study of the possibly additive or synergistic effects of potentially endocrine disrupting compounds.

VIII. CONCLUSION

Since the best-selling novel *OUR STOLEN FUTURE* was published in 1996, millions of people have become acquainted with the term "endocrine disruptor." Yet few people are aware that in that same year the EPA established the EDSTAC to consider how to create a screening and testing program specifically for endocrine dis-

ruptors. Once the EPA has established a screening and testing program for endocrine disruptors, it will need to start conducting risk assessments in order to promulgate drinking water regulation. Today, risk assessment plays a central role in all environmental regulation. Most people would agree that there is a need for government regulations to protect human health from environmental pollutants. It may be premature, however, to focus on endocrine disruptors as such environmental pollutants.

It is simply too early to speculate on the actual human health effects of endocrine disruptors, let alone regulate them as drinking water pollutants, because there are more questions than answers at this time. Additionally, relying on traditional environmental risk assessment as the basis of such regulation could be unduly costly. Traditional environmental risk assessment with its single pollutant approach is probably inadequate for addressing the potentially interactive effects of endocrine disrupting compounds at low levels. If this issue is ignored and drinking water utilities are subjected to stricter regulations, such regulation could be challenged and ultimately invalidated in court. Although some scientists are looking at the consequences of regulating endocrine disruptors as water pollutants based upon traditional environmental risk assessment, more need to be. If not, eventually, some lawyers definitely will.